Wednesday, March 6, 1991
4:00PM–5:00PM, Room 256, West Concourse
Activation Patterns

4:00

PREFERENTIAL PROLONGATION OF TRANSVERSE COMPARED TO LONGITUDINAL CONDUCTION TIME ASSOCIATED WITH LATE POTENTIALS FOLLOWING MYOCARDIAL INFARCTION

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Signal-averaged ECG late potentials (LP) may arise from slowly-conducting myocardium, but the properties of the slowly-conducting tissue are not well characterized. The effect of subacute infarction on conduction transverse (TRANS) and longitudinal (LONG) to ventricular myocardial fiber orientation was studied in 13 dogs 5-12 days after LAD ligation and compared to 7 noninfarcted controls. A 14mm by 14mm multielectrode plaque was sutured to the anterolateral LV epicardium overlying the infarct or normal tissue. Pacing (CL=310ms) was performed from edges of the plaque to obtain LONG and TRANS conduction. Results are expressed as conduction times, not velocities, to avoid assumptions of conduction path.

Results. LONG conduction was similar in infarcted myocardium (2.3±0.9 ms/mm) and in noninfarcted controls (2.0±0.4 ms/mm, p=NS). In contrast, TRANS conduction time was longer in infarcted myocardium (5.1±1.4 ms/mm) than in controls (3.6±0.5 ms/mm, p=0.02). Analysis of variance confirmed a preferential effect of infarction on TRANS compared to LONG conduction (p=.027)

Signal-averaged ECG revealed LP in 9/13 (69%) dogs after infarction. TRANS conduction time in MI dogs with LP (5.9±1.6 ms/mm) was longer than in noninfarcted controls (3.6±0.5 ms/mm, p=0.004), but TRANS conduction time in MI dogs without LP (3.8±0.6 ms/mm) did not differ from controls. In contrast, there was no significant difference in LONG conduction among dogs with or without LP and control dogs.

Conclusions. Conduction time prolongation associated with QRS late potentials after myocardial infarction occurs predominantly in direction transverse to myocardial fiber orientation. The detection of this nonuniform effect on conduction might explain the ability of LP's to identify risk of ventricular tachyarrhythmias.

4:15

AV NODAL WENCKEBACH: NEW OBSERVATIONS ON THE ROSENBLUETH HYPOTHESIS

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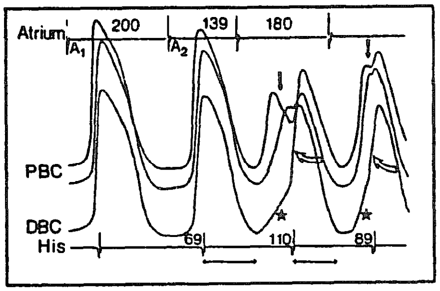
The Rosenblueth hypothesis (RH), in contrast to the decremental hypothesis (DH), explains the Wenckebach phenomenon (W) in the atrioventricular node (AVN) as a development of step-delay at a refractory barrier. Rabbit superfused preparations (n = 10) were used in this study to identify a possible cellular substrate for the RH. Atrial stimulation protocol consisted of 10 basic beats A_1, followed by a conditional A_1A_2 interval, followed by a fixed His-atrial (HA) coupling intervals. Action potential (AP) mapping revealed the existence of specific cells in the N-NH region of the AVN which at shorter coupling intervals qualified as "barrier" cells. A typical example for A_1A_2 = 200 ms and HA = 70 ms is shown below. At the atrial coupling intervals of 139 and 180 ms the proximal barrier cells (PBC) developed a double-hump AP with the first hump amplitude related in a "decremental" cycle-length dependent fashion to the preceding AA intervals (straight arrows). The distal barrier cells (DBC) generated step-delays (stars) representing most of the increment of the AVN conduction time. The step delay was followed by a steeper DBC upstroke synchronized with the His and reflecting backward toward the PBC (curved arrows). A shortening of the DBC AP duration (↔, measured after the His inscription) was apparent after shorter AA intervals modulating the DBC diastolic recovery time.



Thus, the increments in the conduction time depended both on the changes in the PBC AP amplitude and the DBC AP duration. This multifunctional mechanism combines features from both the DH and RH. It can explain the variability of W patterns observed even at fixed atrial pacing intervals.

4:30

EFFECTS OF ELECTRICAL AXIS OF STIMULATION ON THE PATTERNS OF ACTIVATION AFTER SINGLE STRONG PREMATURE STIMULUS IN OPEN-CHEST DOGS

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The graded response hypothesis of ventricular vulnerability predicts that, after a single strong premature stimulus (S_2) is given at a different location than the baseline stimulus (S_1), the area of direct excitation should be located between S_1 and S_2 sites, where intersection between field strength and refractoriness is optimal for activation to occur. This hypothesis does not explain ventricular vulnerability when S_1 and S_2 are at the same site, nor does it incorporate the effects of fiber orientation. To test this hypothesis, computerized mapping studies with 56 closely (2.5 to 5 mm) spaced bipolar electrodes were performed in 6 open chest dogs to determine the patterns of activation after S_2 when S_1 and S_2 were given to the same or different sites on the RV. When S_1 and S_2 were given to the same site, 27 of 57 early sites registered after multiple episodes of S_2 were more than 1 cm away from the site of S_1 and S_2. When S_1 and S_2 were given to different sites, with the line connecting S_1 and S_2 transverse to the myocardial fiber orientation, 21 of the 85 early sites occurred in the area opposite to the site of S_1. In comparison, when the line connecting S_1 and S_2 was parallel to the fiber orientation, none of the 63 early sites occurred opposite to the site of S_1. The intersection between field strength and refractoriness cannot explain these activation patterns. It is hypothesized that, after S_2, the graded response may spread from the S_2 site to area more than 1 cm away. If the spread of graded response is slower than the recovery of excitability, then eventually the graded response will reach fully recovered cells to trigger an activation. This explains why the impulse originates from area away from S_2 even when S_1 and S_2 are given to the same site. When S_1 and S_2 are given to different sites, the recovery of excitability is fastest transverse to fiber orientation. Thus, the earliest activation after S_2 may occur opposite to the S_1 site when the line connecting S_1 and S_2 is transverse to fiber orientation, even though the intersection between the field strength and refractoriness does not favor early sites to occur in that area. Thus, in addition to the intersection between the field strength and refractoriness, the spread of graded response and the effects of fiber orientation on the rate of repolarization are also needed to explain the patterns of activation after single strong premature stimulus.

4:45

IMPACT OF MYOCARDIAL FIBER ORIENTATION ON CELLULAR RESPONSE TO COUNTERSHOCK

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We investigated whether excitation and action potential prolongation (APP), known to occur with defibrillation shocks, depend on the orientation of the potential field with respect to the myocardial fibers. Rabbit papillary muscles (n=7) were bathed in oxygenated Tyrode's solution at 37°C and paced (S1), at 0.5 Hz. Rectangular stimuli of 2 ms duration (S2) were applied from large plate electrodes at opposite ends of the bath in diastole or during the action potential (AP). Recordings of the AP with a glass microelectrode and S2 potential gradient (0-14 V/cm) were obtained for S2 oriented along (L) or across (T) fibers. APP was measured as the difference between the durations at 90% repolarization of each shocked AP and the preceding (control) AP. RESULTS: APP, S1-S2 interval, and time constant of APP (τ) are given as fractions of the control AP duration of 151±32 ms (mean±sd). S2 diastolic excitation threshold and APP for S1-S2=0.7 are tabulated.

	Excitation (V/cm)	APP 4 V/cm S2	APP 8 V/cm S2	APP 14 V/cm S2
		(fraction of control AP duration)		
L	0.68*±0.16	0.20*±0.11	0.36*±0.08	0.36±0.13
T	1.23±0.27	0.04±0.04	0.20±0.04	0.30±0.09

* p<0.05 compared with T

S2 of 2.5 V/cm given during the AP produced no significant APP, rather a new AP when S1-S2>0.9. For 8 and 14 V/cm S2, plots of APP vs S1-S2 increased gradually such that APP closely fit the function $Ae^{(S1-S2)/\tau} + B$ (mean square error<0.007, r>0.95). Time constants, τ, of APP for L and T were 0.23-0.34 of the control AP duration. CONCLUSIONS: 1)Diastolic excitation threshold is approximately half as great for S2 oriented along fibers compared with across 2)For S2 during the AP, 2.5 V/cm S2 produce "all-or-nothing" response, whereas 8-14 V/cm S2 produce a response that increases gradually when S1-S2 is increased 3)APP by S2 of 4-8 V/cm is greater for S2 along fibers than across. APP becomes less dependent on orientation for S2 as strong as 14 V/cm. Thus, fiber orientation may be important for defibrillation with low gradient S2.